2024301280

Burch, P.R.J., "Smoking and Lung Cancer," Lancet, p. 950, October 19, 1974. (SH)

". . . the recorded increases in lung cancer, considered for men and women separately, are not associated in time with the increases in cigarette smoking."

p. 950

Mancuso, T.F. & T.D. Sterling, "Black and White Migrants in the U.S. - A Study of Lung Cancer in Ohio Residents (1959-1967)," Presented at <a href="https://linear.org/length-15/">https://linear.org/length-15/</a> (SH):

"The increase of lung cancer among nonwhite males has raised the question - whether the increase may be related to smoking habits. . . . If the cigarette hypothesis is used then the lung cancer for the white males should be higher than the black. In actual fact, the opposite has occurred, the blacks, nationally in the United States, have a much higher rate. . . "

p. 4

Burch, P.R.J., "Does Smoking Cause Lung Cancer?" New Scientist 611886: 459-463, February 21, 1974. (SH)

"Modern necropsy studies have shown that large errors are still associated with the clinical diagnosis of lung cancer. . . . these and other postmortem studies are the best indicators we have and they make nonsense of the prodigious increases in lung cancer mortality inferred from death certificates."

p. 462

"I am unable to sustain the hypothesis I once held: that lung cancer 'is almost entirely due to cigarette smoking.' At the same time, I am unable to refute Fisher's constitutional hypothesis which offers a plausible and well supported interpretation of numerous otherwise paradoxical findings."

Hickey, R.J., et al., "Aryl Hydrocarbons, Smoking and Lung Cancer," New England Journal of Medicine 290(10): 576-577, March 7, 1974. (SH)

"The association of cigarette-smoking behavior with lung-cancer risk is well known, but inference of causality from correlation is invalid. Moreover, ecologically acceptable animal studies have generally failed to support the hypothesis that cigarette smoking causes lung cancer. An alternate hypothesis, that smoking behavior and lung-cancer risk are influenced by a common cause - the individual constitution or genotype - appears compatible with observed data. Smoking may be symptomatic of constitutional deficiencies that render smokers, on the average, more vulnerable than nonsmokers to damaging effects of air pollutants."

p. 577

Higgins, I.T.T., "Trends in Respiratory Cancer Mortality," Archives of Environmental Health 28: 121-129, March, 1974. (SH)

"A curious feature of the comparison of these countries is that the respiratory cancer death rates of men in England and Wales have levelled off, whereas in the United States they have continued to rise, despite the fact that cigarette consumption appears to be higher in the United Kingdom than in the United States."

p. 127

". . . when absolute levels of consumption in the two countries are compared, it becomes much harder to maintain that the changes in the rates, which have occurred in the United Kingdom, are due to changes in cigarette consumption."

p. 127-128

"The trends in London do suggest, however, that further, more detailed study of mortality in relation to levels and changes in particulate pollution might be rewarding."

Sterling, T.D., "A Critical Reassessment of the Evidence Bearing on Smoking as the Cause of Lung Cancer," American Journal of Public Health 65(9): 939-953, September, 1975.

"If we pull together the information which has become available in the last few years about the prospective studies, we find substantial support for the possibility that the findings linking smoking to lung cancer, and perhaps also to other diseases were due to a faulty selection process that introduced a large number of biases."

pp. 945-946

"Recent findings have verified that lung cancer mortality rates, both in this country and in England and Wales, have stabilized and begun to decline for younger and middle-age population groups. . . . Clearly, it would be unreasonable to observe a decline in lung cancer rates at a time when the consumption of cigarettes is increasing if it were true that cigarettes are a major cause of lung cancer. The parallel observation of the leveling off and decline of lung cancer in this country and in England ought to have far-reaching negative implications."

p. 946

"Lung cancer mortality for migrant populations falls between the rates in country of origin and new host country. This observation has been established predominantly for English immigrants to the U.S., Canada, South Africa, Australia, and New Zealand. . . . The shift in lung cancer deaths from origin to host rates in the immigrating population suggests the importance of environmental factors in the etiology of this disease."

Burch, P. R. J., Problems in the Interpretation of Cancer Statistics with Special Reference to Lung Cancer, Journal Society of Occupational Medicine 25: 2-10, 1975.

"My analysis. . .indicates that most human cancers, as recorded in national mortality statistics, are spontaneous in origin. I infer that they arise, in genetically predisposed persons, as the result of the intrinsic instability of genes in stem cells of the central system of growth-control."

p. 9

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Russek, H. I., Statement, Hearings Before the House Committee on Interstate and Foreign Commerce, June 30, 1964.

"This study (of more than 12,000 persons in 14 occupational groups) revealed that emotional stress appeared to be far more significant than heredity, dietary fat, tobacco, obesity or physical inactivity in the development of heart attacks."

Cederlof, R., et al., "Cardiovascular and Respiratory Symptoms in Relation to Tobacco Smoking - A Study of American Twins," Archives of Environmental Health 18: 934-940, June, 1969.

"It seems that genetic factors are important in the development of coronary symptoms. We believe that along with social, dietary, and other environmental factors, the genetic factors may contribute to the higher prevalence among smokers than among nonsmokers."

p. 940

Seltzer, C. C., Statement, Hearings Before the Committee on Interstate and Foreign Commerce, House of Representatives, April 15-May 1, 1969, pp. 531-545.

"It will be regrettable, if the impact of the prestige of the U. S. Public Health Service led scientists and the public to believe in and accept as firmly established facts which, on the basis of current knowledge, are speculative and lacking in scientific validity. The situation demands not special pleading but scientific truth, namely, what is reasonably established. And, certainly, it has not been reasonably established that cigarette smoking causes coronary heart disease."

pp. 533-534

Evans, W., Statement, Hearings Before the Committee on Interstate and Foreign Commerce, House of Representatives, April 15-May 1, 1969, pp. 1246-1249.

"The incrimination that smoking causes or accelerates heart disease from atherosclerosis of the coronary arteries is wholly unwarranted."

Brown, J., et al., "Nutritional and Epidemiological Factors Related to Heart Disease," World Review of Nutrition & Dietetics 12: 1-42, 1970. (SH)

"A comparative epidemiological study of possibly related factors was made using 1994 middle aged men, including over 500 pairs of brothers, one of whom lives in Ireland and the other in Boston. . . . The proportion of calories derived from fat and saturated fat, the serum cholesterol, the blood pressure levels and the amount of cigarette smoking did not differ markedly. The weight, skinfold thickness and number of abnormal electrocardiograms were higher in the Boston subjects. A study of the pathology of coronaries and aortas from autopsies revealed much earlier serious atheromatous involvement in the Boston than in the Irish specimens."

p. 40

Jenkins, C. D., et al., "Association of Coronary-Prone Behavior Scores with Recurrence of Coronary Heart Disease," Journal of Chronic Diseases 24(10): 601-11, November 1971. (SH)

"Evidence has been accumulating in recent years that social and psychological factors are involved in an important way with the etiology of coronary heart disease.

"This overt behavior pattern (Type A) has been shown to be associated with increased prevalence of coronary heart disease (CHD) by three different research groups. . . "

Jenkins, C. D., "Psychologic and Social Procursors of Coronary Disease," (First of Two Parts), New England Journal of Medicine 284(5): 244-255, February 4, 1971. (SR)

"These limitations in the current knowledge of the etiology and modes of prevention of coronary disease argue for broadening the search for contributing causes and possible dynamics of pathogenesis, rather than merely intensifying the study of the few traditional 'risk factors.'" p. 244

Soloff, L.A., Statement, Hearings Before the Consumer Subcommittee of the Committee on Commerce, U. S. Senate, February 1, 3 & 10, 1972 (as quoted by Senator Marlow Cook, Closing Statement, p. 286). (SH)

"'In conclusion, the data linking heart disease and smoking are still questionable. Studies with particular reference to twins in the United states and Sweden and epidemiological studies in many countries fail to show a correlation between smoking and coronary heart disease. Human studies on smoking that utilize highly artificial circumstances are of questionable validity. Such data do not provide support for the proposed arbitrary limitation on so-called 'incriminated agents' in cigarette smoke.'"

Keys, A., et al., "Probability of Middle-Aged Men Developing Coronary Heart Disease in Five Years," <u>Circulation XLV: 815-828</u>, April 1972. (SH)

"Coronary heart disease (CHD) is now commonly held to have a multivariable causality; in other words, several, perhaps many, variables promote the disease."

p. 815

"It will be noted also that for the three most universally established risk factors, age, blood pressure, and cholesterol the difference between the cases and noncases is larger for hard CHD than for any CHD. This indicates that the more secure the CHD diagnosis, the more clearly is the disease related to age, blood pressure and serum cholesterol."

Werko, L., "The Borderline Between Health and Disease, Prevention or Treatment?" in Skandia International Symposia Early Phases of Coronary Heart Disease, Nordiska Bokhandelns Forlag, Stockholm, 1973, pp. 341-362.

(:

"Even though many would like to interpret the statistical relation of several risk factors to the later development of disease as a causal one, and consequently see the interference with risk factors as the primary measure of prevention, there has so far not been any study showing that changes in--or even removal of--one or several risk factors lead to a decreased incidence of coronary artery disease in a free-living population compared with adequate controls."

p. 350

- ". . .I would suggest that the Gospel from Framingham should not always be accepted as a heavenly truth.
- ". . .It has also become increasing clear that half of the cases of CHD occurring in the U.S.A. cannot be explained by theories involving the current risk factors only. This means that other less known, supposed or completely unknown factors are as important for this disease as all the often discussed risk factors together. Also demonstrating the magnitude of ignorance is the complete lack of explanation for the marked geographical differences in incidence of CHD. . ."

p. 357

"Is it not possible that the present Western society with its emphasis on economic success, high standard of living calculated in monetary units and gadgets is breeding the coronary prone man?"

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Russek, H. 1., "Progress in the Treatment and Prevention of Coronary Heart Disease," American Family Physicians, pp. 68-74, September 1973.

"Even the statistical relationship between smoking and coronary heart disease may reflect the importance of emotional stress rather than cigarettes per se. The report that 100,000 physicians in the United States have given up smoking led us to investigate possible alterations in mortality statistics in this group. . . . It is evident that there has been no increase in the average age at death among physicians during the past 16 years. . . . While it is possible that the full results of this abstinence have not yet been seen, the resolution of underlying stress rather than smoking per se may be the crucial factor. . . These findings are consistent with the apparent predisposition of doctors to coronary heart disease, a vulnerability which can be attributed to the stresses in their way of life."

p. 73

Seltzer, C. C., "More on Smoking and Heart Disease," New England Journal of Medicine 289(22): 1200-1201, November 29, 1973.

"Unless these conflicts in the data are satisfactorily disproved or reconciled, the current enthusiasm for cigarette smoking as a major risk factor in coronary heart disease may become an outstanding fallacy of our era." Fisher, E. R., et al., "Cigarette Smoking and Cholesterol Atherosclerosis of Rabbits," Archives of Pathology 98: 418-421, December 1974.

"Although several studies have claimed that nicotine may augment the atherosclerotic process in this species [rabbit], it is noteworthy that the daily dose of this agent that was employed was equivalent to approximately 175 to 525 cigarettes per day in man; certainly an excessive and unrealistic amount."

p. 421

"The results of the present study which fail to reveal any adverse effect of CS [cigarette smoking] on the structural integrity of the cardio-vascular system in the rabbit with and without induced atherosclerosis. . . "

p. 421

Walker, W. J., "Coronary Mortality: What is Going On?" Journal of the American Medical Association 227(9): 1045-1046, March 4, 1974.

"During the past decade, medical and governmental leaders have proclaimed repeatedly that we are having an epidemic increase in death rate from this [coronary heart] disease.

"The vital statistics of the United States tell a different story! The only meaningful rate is the age-adjusted death rate, which peaked in 1963 and has declined since."

p. 1045

Helmers, C., "Short and Long-Term Prognostic Indices in Acute Myocardial Infarction. A Study of 606 Patients Initially Treated in a Coronary Care Unit." Acta Medica Scandinavica (Suppl.) 555: 86, 1974.

"A history of smoking was no more common in those who died during the first day or during the total hospital period. . . No impaired STP (short term prognosis) has been noted in smokers as compared to non-smokers. . . . The present results were in agreement with these findings. . . . Smoking was no more common in patients who died than in survivors. . . A history of smoking was more common among the survivors than among the deceased. . . In the present study none of these factors (among them smoking) was of significant additional prognostic value, when the most important factors were simultaneously taken into account."

Fisher, E.R., et al., "Cigarette Smoking and Cholesterol Atherosclerosis of Rabbits," Archives of Pathology 93: 418-421, 1974.

". . .a causal role of cigarette smoking to arteriosclerotic heart disease has not been convincingly demonstrated."

p. 418

"The results of the present study. . .fail to reveal any adverse effect of cigarette smoking on the structural integrity of the cardiovascular system in the rabbit with and without induced atheriosclerosis. . . "

"Although several studies have claimed that nicotine may augment the atherosclerotic process in this species [rabbit] it is noteworthy that the daily dose of this agent that was employed was equivalent to approximately 175 to 525 cigarettes per day in man; certainly an excessive and unrealistic amount."

Seltzer, C.C., "Smoking and Coronary Heart Disease in the Elderly,"

American Journal of the Medical Sciences 269(3): 309-315, May-June, 1975

"Age-standardized CHD rates and mortality ratios have been computed from data available in four major prospective cohort investigations of smoking and health. The data examined gave consistent results. For elderly men, there were no appreciable excess of risks of CHD mortality or morbidity among cigarette smokers compared to ex-cigarette smokers and non-cigarette smokers. For elderly women, the CHD rates seemed lower in continuing cigarette smokers than in ex-cigarette smokers. These results obtained from cohort data are concordant with previous analyses of secular data. Among elderly people, the risk of CHD is essentially the same with persistence of cigarette smoking than with its cessation."

p. 309

Seltzer, C.C., "Smoking and Cardiovascular Disease," American Heart 
Journal 90(1): 125-126, July, 1975.

". . . the self-selected group of people who choose to smoke are also more vulnerable to CHD than non-smokers. This hypothesis is supported by evidence indicating that smokers and nonsmokers differ in morphology, physiology, biochemistry, personality, and way of life."

p. 125

Syme, S. L., et al., "Epidemiologic Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii and California: Introduction," American Journal of Epidemiology 102(6): 477-480, 1975.

"It has been observed that among men of Japanese ancestry, there is a gradient in CHD mortality increasing from Japan to Hawaii to California. . p. 477

". . . if the incidence rates from this study confirm that differences in serum cholesterol blood pressure, and smoking do not account for the higher rates of CHD seen among Japanese migrants to the continental United States, then this should provide valuable new insights into the epidemiology of coronary heart disease. Further analyses from this study will pursue the possible effects of obesity and of cultural and dietary change on changing rates of this disease."

"The question of correlation between positive skin tests to tobacco extracts and clinical response to tobacco smoke presents great difficulties. . . . The finding that 12% of normal smokers have positive skin responses to tobacco extract yet continue to smoke, would cast considerable doubt on the relationship of positive weal and flare skin response to the clinical response of the subject to tobacco smoke exposure."

p. 52

# BLOOD PRESSURE

Hrustic, O. & M. Saric, "Odnos Navike Pusenja I Uzivanja Alkohola Prema Visini Arterijskog Krvnoh Tlaka U Skupini Aktivnog Stanovnistva," (Study of smoking habit and alcohol consumption in relation to arterial blood pressure in a group of workers) Arhiv Za Higijenu Rada I Toksikologiju 26(1): 15-22, 1975.

"In a group of 759 male industrial workers aged 20-59 (mean age 37 years) the arterial blood pressure was studied in relation to smoking habit and alcohol consumption.

"The results indicate that there is no association between smoking habit and higher values of arterial blood pressure. On the average, nonsmokers even had a somewhat higher blood pressure than smokers."

"On the other hand the frequency of milk intake was significantly lower in the stomach cancer group compared with the controls. . . . No significant difference was observed with regard to the frequency of intake of other items. . . . No striking association was observed with the habits of smoking and drinking. From these results it is apparent that excessive intake of highly salted foods and the lesser intake of milk are closely associated with the occurrence of stomach cancer."

p. 13

Stell, P. M., "Smoking and Laryngeal Cancer," The Lancet, pp. 617-618, March 18, 1972.

". . . the incidence of laryngeal cancer has remained more or less constant for 70 years—a period in which tobacco consumption and the incidence of cancer of the lung have risen sharply. . . . If cigarette smoke is carcinogenic it seems reasonable to suppose that it should predispose to cancer of the mouth, pharynx, and larynx—but there is no irrefutable evidence that it does so."

p. 617

"Any data showing a correlation between heavy cigarette smoking and laryngeal carcinoma must be interpreted with caution because in the entire population the incidence of laryngeal carcinoma has been remarkably constant; this is even more striking when the incidence of laryngeal carcinoma is compared to the incidence of lung cancer, and to the annual consumption of tobacco and of cigarettes."

p. 618

"My analysis. . . indicates that most human cancers, as recorded in national mortality statistics, are spontaneous in origin. I infer that they arise, in genetically predisposed persons, as the result of the intrinsic instability of genes in stem cells of the central system of growth control."

ULCER

Debas, H. T. & M. M. Cohen, "Effect of Smoking on Gastric Secretion Stimulated by Pentogastrin," The Lancet i: 43-44, January 1, 1972.

"We remain convinced that there is no evidence that either smoking or nicotine causes any significant alteration to the gastric secretory response of normal humans and that the effect of these agents on peptic-ulcer patients has not yet been adequately tested."

p. 44

Cooper, P. & S. H. Tolins, "Relationship Between Smoking History and Complications Immediately Following Surgery for Duodenal Ulcer," The Mount Sinai Journal of Medicine 39(3): 287-293, May-June 1972.

"A cooperative study was conducted by the Veterans Administration with the express purpose of comparing four surgical procedures as a treatment regimen in patients with duodenal ulcer disease. . . The results give no evidence of an association between smoking and complications, nor between smoking and operative mortality, nor between smoking and days to discharge. The most obvious trend is probably predictable, namely patients over 50 have more complications and a higher mortality rate than those under 50."

p. 291

Nomura, A., et al., "Cigarette Smoking and Strokes," <u>Stroke</u> 5(4):483-486, July-August, 1974.

"If the relationship between cigarette smoking and atherosclerotic strokes is not age dependent, it follows that cigarette smoking may not be associated with the atherosclerotic process per se."

p. 486

#### THROMBOSIS

Handley, A.J. & D. Teather, "Influence of Smoking on Deep Vein Thrombosis After Myocardial Infarction," <u>British Medical Journal</u> 3:230-231, July 27, 1974.

"As part of a study of the factors affecting the risk of deep vein thrombosis after myocardial infarction a surprising and unexplained finding was that non-smokers had a significantly higher incidence of thrombosis than cigarette smokers."

p. 230

Doll, R., "Smoking and Deep Vein Thrombosis," British Medical Journal 3: 466, August 17, 1974.

"Finally, we may note that smoking does not appear to be a cause of venous thrombosis."

Marks, P. & Emerson, P.A., "Increased Incidence of Deep Vein Thrombosis After Myocardial Infarction in Non-Smokers," <u>British Medical Journal</u>, pp. 232-234, July 27, 1974.

"The incidence of deep vein thrombosis became 52% in the non-smokers and only 5.4% in the smokers. This difference is again highly significant.

"There was no significant difference in the incidence of deep vein thrombosis between those who smoked a pipe and those who smoked cigarettes (none smoked both). Futhermore there was no positive correlation between the numbers of cigarettes smoked and the incidence of deep venous thrombosis.

"The overall frequency of venous thrombosis after myocardial infarction in this series was similar to that reported by other workers, but in our patients the non-smokers were much more likely to develop deep vein thrombosis after their myocardial infarctions than were the smokers."

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Source: https://www.industrydocuments.ucsf.edu/docs/hlbl0000

#### BIOGRAPHICAL INDEX

## OF AUTHORS

ALLEN, HERBERT B. Lt. M.C. U.S.N.

(page 6/5)

Dermatology service, Naval Hospital Philadelphia, Pa.

only known article, 1973 rebuttal to Harry Daniells' wrinkle theory; scientific, and effective

ALVAREZ, WALTER C. M.D.

(page 8/3)

private practice, syndicated medical columnist Chicago, Illinois

professor emeritus of medicine Mayo Graduate School of Medicine University of Minnesota

AVIADO, DOMINGO M. M.D.

(pages 2/1, 2/2)

Professor of Pharmacology, University of Pennsylvania School of Medicine Philadelphia, Pa.

former consultant to the Council for Tobacco Research long association with pro-tobacco side of controversy

BAIR, W.J.

(page 9/1)

Inhalation Toxicology Section, Biology Department
Pacific Northwest Laboratories division of Battelle Menorial Institute
Richland, Washington

animal studies on inhalation, Atomic Energy Commission grantee; experiment on beagles showed no toxic effects of cigarettes; asked for support from CTR or TI

BAUER, D.R.

(page 2/1)

BERKSON JOSEPH M.D.

(page 8/1)

Chief, Division of Biometry and Medical Statistics The Mayo Clinic

medical statistician, most recent piece on file 1960 proponent of the constitutional theory

BREM, T.H.

(page 10/2)

BRIDGE, D.P.

(page 7/4)

BROWER, L.P.

(page 9/1)

BROWN, J.

(page 11/2)

(deceased)

was associated with the Department of Nutrition Harvard School of Public Health Boston, Mass.

specialist in effects of nutrition and environment versus genetics as cause of disease; cigarettes found not contributors, all other things being equal

BUHLER, VICTOR M.D.

(pages 9/1, 9/2, 10/3)

Pathologist, St. Joseph's Hospital Kansas City, Mo.

publically criticized Auerbach-Hammond smoking dogs

BURCH, PHILIP R.J.

(pages 4/3, 6/4, 6/5, 8/6, 10/9)

The General Infirmary; Professor of Medical Statistics, University of Leeds Leeds, England

former opponent of tobacco, changed opinion (article New Scientist 2/21/74, this report page 10/9) argues for a constitutional interpretation, rejects cause and effect relationship

CARR, DUANE M.D.

(page 8/1)

Professor of Surgery, University of Tennessee College of Medicine Memphis, Tennessee

spoke in industry's behalf, 1969 hearings (4/25) contends that "As of the present date, the cause of lung cancer remains unknown." concerned about misdiagnosis of cancer and related diseases which has led to statistical errors over the years.

CEDERLOF, RUNE Ph.D.

(page 11/1)

Department of Environmental Hygiene The Karolinska Institute Stockholm, Sweden

extensive studies on twin pairs show that personality not smoking is probable cause of disease

COAN, RICHARD W. Ph.D.

(page 4/4)

Professor of Psychology University of Arizona Tucson, Arizona

proponent of the constitutional theory

COOPER, DAVID A. M.D.

(page 10/1)

(deceased July 6, 1970)

was associated with University of Pennsylvania School of Medicine Philadelphia, Pennsylvania

specialist in lung cancer; believed fact that nonsmokers contract lung cancer disproves causation theory.

COOPER, PHILIP M.D.

(page 12/2)

Department of Surgery
The Bronx Veterans Administration Hospital
Bronx, New York

limited studies, post-operative effects of smoking on duodenal ulcer patients

DEBAS, H.T. M.D.

(page 12/2)

Faculty of Medicine, University of British Columbia Department of Surgery, Vancouver General Hospital Vancouver, British Columbia, Canada

surgeon; single article

DIAMOND, J.L.

(page 2/3)

Wellington, New Zealand

single article; concerned with chronic bronchitis as a psycho-physiological disorder suggests that it is class-related in Britain.

DOLL, SIR RICHARD M.D. D.Sc.

(page 12/3)

Regius Professor of Medicine The Radcliffe Infirmary Oxford, England

medical statistician; co-author of landmark retrospective study on British doctors, still used as basis of modern investigations, quoted in U.S. Surgeon General reports

ECKHARDT, ROBERT E. M.D. Ph.D.

(page 7/3)

Director, Medical Research Division Esso Research & Engineering Co. Linden, N.J.

specialist in occupational cancers and industrial medicine; particularly concerned with toxic effects of CO

EISENBUD

(page 3/1)

Professor, Institute of Environmental Medicine New York University Medical Center New York, N.Y.

specializes in effects of air pollution, carbon monixide; feels cigarette smoke is the major contributor to high rates of CO concentration in urban air

EVANS, WILLIAM M.D., D.Sc.

(page 11/1)

Consulting Physician to the Cardiac Department of London Hospital, to the National Heart Hospital, and to the Institute of Cardiology London, England

most recent materials 1966; suggests that smoking as cause is only a "wild guess"

EYSENCK, H.J. Ph.D., D.Sc.

(page 4/1)

Director of the Department of Psychology Institute of Psychiatry London, England

his landmark book, Smoking, Health and Personality is basic to constitutional theory of disease causation

#### FEINSTEIN, ALVAN R. M.D.

(pages 8/6, 10/6)

Professor of Medicine and Epidemiology, Yale University School of Medicine Chief, Clinical Biostatistics and Co-Operative Studies Program Support Center, Veterans Administration Hospital New Haven, Conn.

medical statistician; prime spokesman for "detection bias" theory alleging that lung cancer is observed in smokers because it is sought more vigorously in them than in nonsmokers

## FIORENTINO, MARCO M.A.

(page 10/1)

Associate Epidemiologist, Department of Epidemiology University of Texas; M.D. Anderson Hospital and Tumor Institute Houston, Texas

most recent material 1969; medical statistician specialist in age-at-death lung cancer statistics

# FIFER, WILLIAM R.

(page 3/1)

St. Louis Park Medical Center Executive Health Program Minnesota

suggests regular tests for early detection of health problems

#### FISHER, EDWIN M.D.

(page 10/3)

Shadyside Hospital University of Pittsburgh Pittsburgh, Pa.

CTR grantee; specialist in animal experiments denies atherosclerotic effect of nicotine in "realistic" doses

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## FISHER, SIR RONALD A.

(page 11/5)

(deceased)
was associated with Cambridge University
Cambridge, England

"generally regarded as the greatest theoretical statistician of the modern era" (Lancet), although opposed to cigarettes, he objected to the simplistic approach of assuming causality from association

## FURST, ARTHUR Ph.D.

(page 1/2)

Director of the Institute of Chemical Biology University of San Francisco San Francisco, Cal.

CTR grantee, former CTR consultant 29 years in cancer research specializing in chemotherapy; first scientist to be concerned with effects of trace metals in development of cancer

#### HANDLEY, ANTHONY J.

(page 12/3)

Queen Mary's Hospital London, England

single study, 1974, found nonsmokers healthier than smokers with respect to deep vein thrombosis

#### HARDY, JANET B.

(page 6/6)

Department of Pediatrics Johns Hopkins Hospital Baltimore, Md.

Director of the Collaborative Perinatal Mortality Study; found low-birth weight babies of smokers as healthy at ages 1,4, and 7 as children of nonsmokers

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HARKE, H.P.

(pages 7/1, 7/3, 7/3)

Research Institute of the Tobacco Industry Hamburg, Germany

specialist on effects of "passive smoking;" studied smokers and nonsmokers in closed spaces; found no serious effects; suggests that filters greatly reduce effects

HERROLD, KATHERINE M.

(page 10/7)

National Cancer Institute, Laboratory of Pathology Bethesda, Md.

seeks standard classification system for cancers to eliminate statistical errors

HICKEY, RICHARD J. Ph.D.

(pages 4/4, 6/2, 6/5, 8/4, 9/2, 10/10)

Management and Behavioral Science Center The Wharton School, University of Pennsylvania Philadelphia, Penn.

medical statistician; specialist in effects of air pollution

HIGGINS, IAN T.T. M.D.

(page 10/10)

Department of Epidemiology University of Michigan School of Public Health Ann Arbor, Michigan

AMA-ERF grantee, 1967; specialist in effects of air pollution

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HIRIYAMA, TAKESHI M.D., M.P.H.

(page 12/1)

Chief, Epidemiology Division National Cancer Center Research Institute Tokyo, Japan

specialist in effects of air pollution

HUEPER, WILHELM C. M.D.

(page 3/3)

Chief of Environmental Cancer Section National Cancer Institute
Bethesda, Md.

Native of Germany; specialist in pathology, occupational hazards, and carcinogenic chemicals

ITO, HARUO M.D.

(page 2/1)

Professor of Pharmacology Kanagawa Dental College Yokosuka, Japan

CTR grantee; worked with Aviado at University of Pennsylvania on pulmonary emphysema and smoke; found no cause-and-effect relation on rats

JAMES, WILLIAM H. M.D.

(page 6/4)

The Galton Laboratory
Department of Human Genetics and Biometry
University College
London, England

specialist in smoking effect on pregnancy and early childhood development; tends to support Yerushalmy concerning the emotional nature of the smoker affecting the child before and after birth

JENKINS, C. DAVID Ph.D.

(pages 4/3, 11/2, 11/3)

Division of Psychiatry, Department of Behavioral Epidemiology Boston University School of Medicine Boston, Mass.

supports constitutional theory; associates psychic health with disease; supports Type A theory, worked with Rosenman and Friedman

JOHNSTONE, FRANK M.D.

(page 6/7)

Department of Obstetrics and Gynecology Aberdeen Maternity Hospital Aberdeen, Scotland

studied birth weights; found family history predisposes sisters to have low-birth-weight babies, whether or not they smoke

KEYS, ANCEL Ph.D.

(page 11/3)

Laboratory of Physiological Hygiene University of Minnesota School of Public Health Minneapolis, Minn.

accepts smoking as contributor, but not sole cause of coronary artery disease

KISSEN, DAVID M. M.D.

(pages 4/1, 10/1)

(deceased February 1968)
was Director of Psychosomatic Research Unit
Southern General Hospital
Glasgow, Scotland

supported constitutional theory; believed emotional factors important in disease causation; considered to be the pioneer in application of psychosomatic research to practical medicine

#### KOTIN, PAUL M.D.

(page 10/2)

Director, National Institute of Environmental Health Science Research Triangle Park, S.C.

suggests development of "less hazardous cigarette" (appeared at Senate Committee hearings 1967)

# KUTTY, M. KANNAN

(page 10/6)

Institute for Medical Research Kuala Lumpur, West Maylasia

suggests geographic causes of cancer; found no significant correlation between lung cancer and smoking in any of the separate groups in study (Chinese, Malays, Indians)

#### LANGSTON, HIRAM T. M.D.

(pages 8/1, 10/2, 10/8, 10/8)

Professor of Surgery University of Illinois College of Medicine Chicago, Illinois

(note: A.B. and M.D. from University of Louisville)
proponent of thesis that major increase in new cases of lung
cancer comes from people born 1980 to 1900 and that when they
eventually die, the increase will disappear; also cites sex
differences male/female in cancer incidence as negating the
causal relationship

#### LAVE, LESTER B.

(page 3/2)

Professor of Economics: Graduate School of Industrial Administration Carnegie-Mellon University

specialist in statistics; suggests that association of chronic health effects are difficult to associate with either smoking or air pollution; feels air pollution is substantial contributor to mortality in certain areas; suggests smoking alone is probably not sufficient

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LEES, THOMAS W. M.D.

(pages 8/2, 10/4)

Pathologist, Provincial Laboratory Charlottown, Prince Edward Island, Nova Scotia

known for "wave theory" which suggests that the frequency of death from particular diseases rises and falls in successive waves over the centuries; suggests U.S. will peak in 1979; acted as pro-tobacco witness in Canadian Parliament hearings:

LESHAN, LAWRENCE L. Ph.D.

(page 4/1)

Institute of Applied Biology New York, N.Y.

psychologist; suggests connection between emotional life history and susceptibility to cancers

LEVINE, EDWIN RAYNOR M.D.

(page 2/1)

Director, Department of Respiratory, Cardiac and Pulmonary Laboratories Edgewater Hospital Chicago, Illinois

specialist in chest diseases; testified in 1969 hearings; in 1973 spoke out against Administration cutbacks in spending for health research asserting case is not closed

LEWIN, R.

(page 10/8)

# LIGHTFOOT, N.F.

(page 7/4)

Surgeon Lieutenant Institute of Naval Medicine Alverstoke, England

specialist in effects of CO on nonsmoker

#### LUDWIG, JOHN H.

(page 8/3)

Assistant Commissioner, Office of Science and Technology National Air Pollution Control Administration Environmental Health Service (division of National Institutes of Health)

specialist in effects of air pollution

#### MALHOTRA, S.L.

(page 10/5)

Medical Department South Eastern Railway Calcutta, India

studied effects of tobacco on heart patients

#### MANCUSO, THOMAS F. M.D.

(pages 3/2, 10/9)

Research Professor, Department of Occupational Health University of Pittsburgh Graduate School of Public Health Pittsburgh, Pennsylvania

CTR grantee; specialized in environmental, occupational, and ethnic causes of cancer; detailed study of blacks and whites who migrated from rural south to industrial north

#### McCALL, M.G.

(pages 3/1, 10/6)

Department of Medicine Raine Medical Statistics Unit University of Western Australia

specialist in environmental/industrial causes of disease, studied death rate of immigrants to Australia from Britain

NOMURA, ABRAHAM M.D., M.P.H.

(page 12/3)

Epidemiologist, School of Hygiene and Public Health Johns Hopkins Hospital Baltimore, Md.

studied effects of tobacco on cerebral vascular thrombosis, notes no correllation

## O'DONNELL, ROBERT D.

(page 7/2)

Space Biology Laboratory
Medical Center, University of California
Los Angeles, Cal.

toxicologist; concerned with effects of CO on psychomotor performance

#### OKUN, RONALD M.D.

(page 1/1)

Chief, Department of Clinical Pharmacology Cedars-Sinai Medical Center Assistant Professor of Medicine and Pharmacology, University of California Los Angeles, California

concerned over flaws in medical statistics; testified in 1969 hearings

OSER, BERNARD L. Ph.D.

(page 8/7)

Food and Drug Research Laboratories, Inc. New York, N.Y.

medical statistician; concerned over misuse of statistics to achieve desired results

OSTER, KURT A.

(page 8/4)

Pharmacologist and Chief of Cardiology Park City Hospital Bridgeport, Conn.

criticizes use of statistics in Framinghan study; heart specialist; suggests an enzyme in cow's milk might be responsible for atherosclerosis

PETTERSSON, FOLKE

(page 6/6)

Department of Obstetrics and Gynecology, Department of Social Medicine University Hospital.
Uppsala, Sweden

specialist in causes of perinatal mortality

RAE, GORDON

(page 4/4)

Department of Educational Psychology Aberdeen College of Education Aberdeen, Scotland

supports constitutional theory

RIGDON, R.H. M.D.

(pages 1/2, 10/3)

Professor of Pathology University of Texas, Medical Branch Galveston, Texas

testified at 1969 hearings; asserts only a statistical relationship between smoking and lung cancer; supports constitutional theory

ROBERTS, KATHLEEN E. M.D.

(page 2/2)

Private practice Owego, N.Y.

believes that only associations of smoking with disease are political and statistical

ROSENBLATT, MILTON B.

(pages 2/2, 2/3, 8/2, 8/3, 8/5, 10/5)

(deceased January 1975) was attending physician, Doctor's Hospital, New York

specialist in lung disorders; concerned over inaccuracies in autopsy statistics, death certificates etc.; leading proponent of theory that faulty statistics are cause of increase in certain diseases

RUSSEK, HENRY I. M.D.

(page 11/4)

Saint Barnabas Hospital New York

heart specialist; believes stress is cause of disease; asserts that while prevention techniques have improved, stress has increased, and therefore disease rates remain the same; has testified on behalf of industry (not 1969):

### SCHIEVELBEIN, H.

(pages 7/5, 8/5)

Institute for Clinical Chemistry and Clinical Biochemistry University of Munich, Department of Preventive Medicine Munich, West Germany

former head of Research Institute of the German tobacco industry; specialist in "passive smoking," denies detrimental effect on nonsmoker under realistic conditions

SELTZER, CARL C. Ph.D.

(pages 4/3, 5/1, 11/1, 11/4)

Senior Research Associate, Biological Anthropology Harvard University School of Public Health Cambridge, Mass.

long positive association with industry; most recent reports 1975 concerning old age and smoking; concerned with stress; has testified for industry

SELYE, HANS M.D., Ph.D., D.Sc.

(page 5/1)

Director and Professor, Institute of Experimental Medicine and Surgery University of Montreal Montreal, Canada

the leading authority on stress; suggests that if smoking relieves stress for an individual, then it has positive value

SHERMAN, JANETTE M.D.

(pages 2/3, 3/5)

Internist Detroit, Michigan

specialist in occupational hazards; directed study for Health Research. Group (a non-profit organization financed by Ralph Nader's Public Citizen, Inc.) on workplace dangers; asserts over-emphasis on cigarettes to avoid more expensive safety measures

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SOLOFF, LOUIS A. M.D.

(pages 1/2, 11/3)

Professor of Medicine (chief of cardiology, emeritus)
Temple University
Philadelphia, Pa.

heart specialist; presented at June 1975 Maxwell seminar, and at 1970 New York Security Analysts meeting on behalf of industry; asserts no conclusive evidence of causation.

STELL, P.M.

(page 12/1)

Ear, Nose, and Throat Infirmary Liverpool, England

specialist in laryngeal cancer; admits association of smoking with disease, but denies causation because if smoking were damaging, then throat and bronchial cancers should be much more prevalent

STERLING, THEODOR D. Ph.D.

(pages 3/3, 3/4, 9/2, 10/7)

Professor, Faculty of Interdisciplinary Studies Simon Fraser University Burnaby, B.C., Canada

believes association of smoking with lung cancers an "ingenious oversimplification of a complex set of relationships" (statement April 1975); on record opposing government limits to "tar" and nicotine content suggesting that OSHA act demonstrates government belief in other potential causes of lung disease; concerned with ethnic and geographical causes of cancer

STEWART, RICHARD D. M.D.

(pages 3/5, 7/1, 7/4, 7/5)

Department of Environmental Medicine Medical College of Wisconsin Allen Bradley Medical Science Laboratory Milwaukee, Wisc.

specialist in effects of carboxyhemoglobin (COHb); most recent article on COHb and smoking donor's blood on patient

SYME, S. LEONARD Ph.D.

(page 4/2)

Professor of Epidemiology University of California School of Public Health Berkley, Cal.

statistician, social epidemiologist; believes social factors, personality, and stress are causative agents; expands on constitutional theory

WALKER, WELDON J. M.D.

(pages 2/4, 11/5)

Director, Cardiopulmonary Laboratory White Memorial Medical Center Los Angeles, Cal.

concerned with misuse of medical statistics as misleading; opposed to tobacco subsidies

WALTER, E.

(page 4/2)

YAGLOU, C.P.

(page 7/1)

Professor of Industrial Hygiene Harvard University School of Public Health Cambridge, Mass.

only paper 1955, landmark study on ventilation requirements; denies health hazard of smoking on nonsmokers

YERUSHALMY, JACOB Ph.D.

(pages 6/1, 6/2, 6/3)

(deceased)
Professor, Division of Biostatistics
University of California School of Public Health
Berkley, Cal.

leading statistician; specialist in perinatal mortality; asserts that while infants of smokers are smaller, they are no less healthy; believes that it is the smoker and not the smoking that causes problems; proponent of the constitutional theory